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Tracheal Stenosis

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The increasing use of endotracheal intubation in support of various surgical methods has resulted in more injury to the tracheal mucous membrane than was formerly supposed. Tracheal injury may result in fibrosis and narrowing of the trachea. When tracheal stenosis is of a sufficient degree to necessitate repeated bronchoscopies and dilations, the patient's time under medical care and total morbidity may be considerably lessened by aggressive approach, involving resection of the diseased portion of the trachea.

TRACHEAL STENOSIS, as seen today, is largely a "disease of medical progress." With the advent of improved, more aggressive management of airway and pulmonary problems with various types of intubation devices has come a brand new complex of assorted problems. In the past, tracheal stenosis was an infrequently seen lesion associated with rare tumors, pulmonary tuberculosis, and the problem of extrinsic compression. As the philosophy of management of thoracic injuries changed, and as cardiac surgery came into its own, more and more patients were subjected to tracheal intubation. From among them has come a small but significant group of patients with tracheal injury secondary to intubation which has led to tracheal stenosis. Although intubation is the direct cause of the lesion, there are multiple factors connected with the intubation that contribute to the development of the lesion.

Much work[1-6] has been done to define predisposing factors in an attempt to improve management and reduce the incidence of strictures. Early conservatism in treatment has given way to an aggressive philosophy with a gratifying improvement in subsequent results. The purpose of this presentation is to describe three illustrative cases and to review the current etiologic concepts and management, as well as the results of treatment of this lesion.

Case 1. A 33-year-old white man was admitted to the Santa Clara Valley Medical Center emergency room on 5 April 1969 with a nine-hour history of severe substernal chest pain radiating to both arms, associated with nausea and diaphoresis. The patient's father had died at age 41 of a myocardial infarct, as had one uncle, also at age 41. Cardiac arrest occurred while the patient was in the emergency room, requiring defibrillation, external cardiac massage, and tracheal intubation. An electrocardiogram confirmed an acute myocardial infarction. Following successful resuscitation from the arrest, the patient was comatose, unresponsive and had no spontaneous respirations. On 6 April, elective trache-

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ostomy was carried out under local anesthesia in the operating room. The tracheal incision extended from the second to the fourth tracheal rings. A No. 9 tracheostomy tube was inserted without difficulty. The patient made a slow recovery from cardiac arrest, with a stormy course complicated by pneumonitis and seizure activity. The patient required assisted respiration until 19 April, after which spontaneous respirations were adequate. The tracheostomy tube was removed 9 May 1969 after 35 days of tracheal intubation. Cultures from serial tracheal aspirates revealed a hemophilus species, alpha and beta streptococcus, candida albicans, bacteriodes, diphtheroids, neisseria, and E. coli. Following removal of the tracheostomy tube, the patient was transferred to the Rehabilitation Service for management of cerebral impairment. He made slow and incomplete recovery from the anoxic insult but improved enough for transfer to a nursing home. There he did well until 30 August 1969 when progressive shortness of breath developed, with wheezing. Transferred to the emergency room on 31 August 1969, he was treated for a bronchospasm without relief of symptoms. An x-ray film of the chest showed tracheal stenosis. While in the emergency room, the patient's condition deteriorated rapidly, and attempts at oral and nasal intubation of the trachea were unsuccessful. Tracheostomy was performed in the emergency room and tight stenosis was encountered at the cuff site. This was dilated bluntly with a clamp until a 6.5 mm uncuffed tube could be passed. Respiratory distress was immediately relieved. Over the next 72 hours, the stricture dilated rapidly and required progressively larger tubes for occlusion of the airway around the tube.

At subsequent bronchoscopic examination some mucosal ulceration, but no persisting stricture was observed. The tracheal tube was removed and the skin allowed to close. Tomography on 30 September 1969 showed no recurrence of stenosis. The patient did well until 13 October 1969 when shortness of breath again prompted an emergency room visit, and he was handled with symptomatic therapy. He returned the next day with continuing complaints of shortness of breath, and a tracheogram at this time revealed recurrence of the stricture.

Tracheostomy was again performed, this time in the operating room, and the stricture dilated. After appropriate preparation, the patient was taken to the operating room on 24 October 1969 and underwent a tracheal resection with a primary anatomosis via a median sternotomy incision. His postoperative course was uneventful, and the tracheostomy tube was not re-inserted at the conclusion of the procedure. Six weeks later bronchoscopic examination was repeated, and satisfactory healing of the suture line, with no recurrence of stricture, was noted.

Current follow-up at one year postoperatively reveals no recurrence of the stricture, and the patient has had no further respiratory difficulties. However, his mental state continues to be suboptimal.

Case 2. The patient, a 42-year-old woman, was admitted for the first time 6 May 1970 with chief complaint of tracheal stenosis. She had been in a state of good health until January 1970, when she took a massive overdose of glutethimide (Doriden) and was treated at another hospital. She was comatose for two weeks and tracheostomy and assistance with respiration were necessary. After recovery she was found to have tracheal stenosis at the level of the sternal notch. Tracheostomy was done again, just below the stenotic area, at another hospital in March 1970. Over the following two months she had tracheal dilation four times with passage of an 8 mm bronchoscope, which consistently showed narrowing down to 3 or 4 mm. A post-dilatation tracheogram showed a 1.5 cm segment of stenosis just above the sternal notch and just below the area of the last tracheostomy. Even with the tracheostomy tube in place, the patient complained of shortness of breath and wheezing, which worsened with activity and talking. She did her own tracheal care at home, where she lives with her husband and a six-year-old daughter. She was referred for definitive treatment. She appeared in no acute distress at the time of admittance. Blood pressure was 105/75 mm of mercury and respirations were 22 per minute and slightly labored. A tracheotomy tube was in place. Head and neck examination were within normal limits except for the tracheostomy. Breath sounds were clear on both sides of the chest. The breasts were normal. Heart rate and rhythm were normal and there were no murmurs. The abdomen was soft and without masses or tenderness. Rectal-pelvic examinations and the remainder of the physical examination were within normal limits. The impression was tracheal sten-

osis secondary to tracheostomy tube after an overdose of Doriden. X-ray studies of soft tissues of the neck did not reveal the area of stenosis, which was believed to be at the sternal notch. No abnormality was noted on an x-ray film of the chest. An electrocardiogram was normal. Complete blood count was within normal limits. The hematocrit was 43. Urinalysis was normal. Chemistry screening battery was within normal limits, as were electrolytes. On 7 May 1970 the tracheostomy tube was removed without immediate difficulty to the patient but within three hours she was having increasing respiratory difficulty with retraction, and the tracheostomy tube was replaced. On 11 May resection of the trachea at the site of the stoma and stenosis was carried out. Bronchoscopy showed only a 1 to 2 mm opening above the tracheostomy tube. Postoperatively the nasal endotracheal tube was kept in place for two days and then removed. The patient did well and the wound healed nicely. On 16 May 1970 she was discharged. Her talking and breathing were within normal limits at this time. Four months later the patient was well.

Case 3. The patient, a 19-year-old boy, was readmitted to the Stanford Medical Center 31 January 1970, after having been discharged three days earlier with a diagnosis of upper respiratory obstruction. A long endotracheal tube had been used four weeks earlier in treatment for drug ingestion, and tracheal stenosis had developed. The patient had first been admitted 21 January 1970, and bronchoscopy then had shown the stenosis. The area was reamed and a tracheal stent inserted. When the patient was readmitted because of re-obstruction, the T tube was found to be plugged with secretions. On 5 February he was taken to the operating room, and when bronchoscopy showed re-stenosis of the area, another 12 mm tracheal stent was inserted. On 13 February the patient was sent home, where he had a suction apparatus to take care of the stent. Nevertheless, he had increasing trouble with secretions and blocking of the upper airway. He was readmitted 24 February 1970, with complaint of difficulty in breathing, for definitive treatment of the tracheostenosis. Tomograms of the trachea showed stenosis for approximately 3 to 3.5 cm above the stoma of the tracheostomy. On 28 February indirect laryngoscopy was carried out. Granulation tissue, completely occluding the trachea in the sub-glottic region, was seen. The tube was not seen. On 9 March four rings of the trachea (approximately 5 cm) was resected. The postoperative course was ordinary. The patient's neck was kept in a flexed position with the chin down. Cloxacillin was administered until 16 March and the patient remained afebrile. Postoperative x-ray films of soft tissues of the neck showed full diameter at the site of tracheal anastomosis. The patient was discharged 30 March and at last report was working and had no difficulty in breathing.

Etiology

The etiology of tracheal stenosis can be divided into several broad categories. Tracheal intubation, external trauma, tumors, and tracheomalacia are the most important sources of damage to the trachea. Tracheomalacia is usually due to extrinsic compression seen with large goiters, (particularly the substernal type), to congenital vascular rings, and to other mediastinal tumors that produce prolonged direct pressure on the trachea. The tracheomalacia with expiratory collapse seen in the patient with chronic obstructive pulmonary disease is a cause of a different kind, and the methods of treatment are considerably different.

Intrinsic tumors of the trachea are quite rare but can produce a clinical syndrome indistinguishable from a stenotic scar. The prototype tumor is a cylindroma of the trachea which probably is a low grade adenocarcinoma. Some 20 histologic types of tumor have been described, but there are basically four main categories. These present a wide spectrum of degree of malignancy, but many of these are low grade and lend themselves well to local resection for cure.

External trauma to the trachea in the form of blunt or penetrating injury can lead to stenotic lesions that require subsequent therapy. Chemical injuries such as may be caused by smoke inhalation, aspiration of gastric contents, or aspiration of ingested caustic solutions have not been described as a cause of localized tracheal stenosis, although severe diffuse superficial injury may occur. The use of tracheal intubation is the most common, most preventable cause and the lesion produced is probably the most easily treatable. The use of tubes in the trachea can cause damage regardless of the site of entry—nasal tracheal,

oral tracheal, or tracheostomy. Stenosis may follow intubation for even so short a time as 36 hours, but usually it is associated with longer periods.

Infection in the airway is another prominent contributing factor. In spite of fastidious local care and constant reminders to the nursing staff about the necessity for sterile technique, in virtually all cases cultures of material taken from a tracheostomy wound grow infective organisms. The patients are often receiving antibiotics that eliminate the local flora and permit the appearance of an antibiotic-resistant opportunistic invaders. In a specially designed and carefully run respiratory care unit, Gibson[6] reported 154 consecutive patients requiring respiratory support. One hundred and seven had cultures, of which only 12 were sterile. The most common organisms cultured were E. coli, Friedlander's bacillus, staphylococcus, pseudomonas, and candida albicans. Of 96 survivors, ten had ulcerated lesions of the trachea with positive cultures in the tracheal aspirate. Clinically significant tracheal stenosis developed in all ten of these patients.

Physical characteristics of the tracheostomy tubes also play a role in the development of tracheal lesions. Excessively large tubes, high pressure in the tube cuff, and the chemical composition of the tube are all potential sources of trouble. It is clear that high cuff pressures will produce a local ischemic lesion in the relatively rigid trachea, leading to ulceration of the trachea, with erosion and subsequent destruction of the cartilagenous support. Specially designed low pressure cuffs with broad bearing on the tracheal wall produce essentially no damage to the respiratory epithelium even with prolonged constant cuff inflation. Portex endotracheal tubes used to contain an irritative chemical that sometimes produced stenotic lesions with both tracheal and urethral intubation. The composition of these tubes has since been changed, and since then no such lesions have been ascribed to them.

Diagnosis

The diagnosis of tracheal stenosis is relatively simple if the possibility is considered, as it should be if there is history of tracheostomy or prolonged nasal tracheal intubation. The only major difficulty is that patients remain asymptomatic, with no physical signs, during the early phases of tracheal narrowing. But when the stricture reaches a critical stage symptoms may become acute shortness of breath, inability to clear secretions, even sudden death. History, other than that of previous tracheal intubation, is generally not helpful. Wheezing may be heard in the central portion of the chest or in the neck. This may occur only on expiration or with forced expiration. Probably every significant stricture will have some audible wheeze in the neck. Posterior-anterior and lateral x-ray films of the neck with soft tissue technique will generally show the narrowed air column with good detail. Contrast studies with tantalum powder or aqueous lipiodal will give further information. Bronchoscopy will reveal the lesion without difficulty, but with monocular vision it is difficult to determine how long a segment is damaged. Bronchoscopy is primarily indicated to evaluate the status of the remainder of the tracheobronchial tree and to assess the pliability of the strictured area. Even though the bronchoscope may easily pass through the stricture, if the procedure is done under local anesthesia, forced expiration or cough will reveal so decided a narrowing of the lumen that the airway is inadequate.

The dilating effect of bronchoscopy may produce temporary relief of the airway obstruction, but this is generally short-lived, and the improvement should not lull the physician into a false sense of security. Serial bronchoscopy for dilatation or prolonged close follow-up is necessary in the management of these patients.

Treatment

As with many other problems, this lesion is better prevented than treated. Many ingenious tracheostomy tubes have been devised with this in mind. Tubes with oversized cuffs designed to produce a more diffuse and lower pressure on the tracheal wall have been used. The disadvantage of these tubes is that the redundant cuff can slip over the end of the tube and close it completely. Devices that trigger cuff inflation by the respirator so that balance is maintained between the pressure in the cuff and in the airway have been used. Since the cuff deflates cyclically during expiration, it presses only intermittently on the tracheal wall. This has theoretical appeal, but it eliminates the protection of the inflated cuff against aspiration of swallowed or vomited ma-

terial. In addition the mechanisms are complex and mechanical failures sometimes occur.

As was mentioned earlier, bronchoscopy can give temporary relief of symptoms, and some patients can be successfully managed over long periods by repeated bronchoscopy, but this requires close cooperation by the patient, close observation, and elasticity at the point of stricture.

Internal stenting with reinforced silastic T-tube stents has been used at Stanford Hospital with moderate success. These tubes are placed in the trachea following dilation of the stricture with the right-angle portion extending through the tracheostomy stoma. These can be used for stoma or cuff level strictures. The tubes are generally left in place for nine months and then removed. They require close early supervision, as secretions may become a problem. With long-term use, secretions become less troublesome. However, exuberant granulation tissue stimulated by the presence of a foreign body can lead to narrowing of the trachea or occlusion of the tube. Another hazard is displacement of the tube, with occlusion of the airway ensuing. Some patients tolerate the tubes well and retain them in appropriate positions to a successful conclusion of their stenosis problem. Others have major local problems which require termination of this form of treatment.

Surgical resection of the stenotic lesion is the treatment of choice for localized fibrous strictures if the narrowed segment cannot be dilated easily or stent insertion is poorly tolerated by the patient. Short areas of trachea can be resected and primary repair done with relative ease. The surgical approach may be from the neck, the mediastinum or the right side of the chest, depending upon the position and length of the stricture. Thus far, prosthetic replacement has been uniformly successful. Synthetic material does not produce an adequate functioning trachea in that secretions are poorly mobilized, and healing between the prosthesis and the trachea generally fails. The only limiting factor in resection of the trachea is the length of the lesion. It is important to carry the resection back to normal trachea, above and below, for good results without recurrence.

Grillo,[7-11] in extensive studies to determine the maximum length of trachea that can be resected with primary repair, demonstrated that tracheal anastomosis with greater than 1700 grams of tension will separate or will heal by scarring that makes a new stenotic lesion. Various maneuvers can be used to obtain length to facilitate a tension-free anastomosis. The average length as determined by cadaver studies is 11 cm. "Lowering the trachea" by release of the suprathyroid attachments as described by Dedo and Fishman[3] (2 to 3.5 cm); flexion of the neck (4 to 5 cm); mobilization of the right hilum with division of the pulmonary ligament (3.5 to 5 cm); division of the left mainstem bronchus with reinsertion into the bronchus intermedius; freeing the pulmonary vessels from the pericardium; and advancement of the cervical trachea on a vascular pedicle from the inferior thyroid artery (4 cm) have all been described. These methods need be used only for relatively long strictures or when the mediastinum must be avoided for some reason, such as previous cardiac operation. In the usual short stricture, local mobilization of the strictured area of the trachea combined with flexion of the neck is all that is required for adequate length to permit tension-free anastomosis.

Results

In general, in the very small series reported the best results are with primary repair. Only Grillo's series is big enough to warrant conclusions. Use of a T-tube stent is not widely reported, and results at Stanford have not yet been published. No one has described a case using a combination of laryngeal lowering and extensive intrathoracic mobilization in the same patient. Only four cases of the laryngeal lowering have been reported, and in all of them the patients are doing well. Combining six published series, the following results were compiled:

		Results			
Procedure	*No. of Patients*	*Good*	*Satisfactory*	*Poor*	*Deaths*
Dilation	13	4	7	2	0
Resection	44	32	5	7	3

Undoubtedly many cases successfully treated by dilation alone have not been reported. It is probable that a higher proportion of operative cases are reported, for the literature is still so scant that reports are acceptable for publication. Therefore it is a reasonable assumption that the results listed above may be considerably biased

and that they do not reflect the true picture. At the Santa Clara Valley Medical Center in the past ten years there have been seven patients with established diagnosis of tracheal stenosis. Five were managed with endoscopic removal of granulation tissue or dilatation, one by permanent tracheostomy, and one by resection (presented herein). All these patients are doing well, although two of them have been lost to follow-up.

Conclusions

Tracheal stenosis is an uncommon lesion largely occurring secondary to tracheal intubation with respiratory support. Contributing factors are multiple, but the final common pathway is a localized destruction of tracheal wall with subsequent healing by fibrosis that causes a stenotic lesion as contracture occurs. There are several well-established means of treatment, with results generally satisfactory if the diagnosis is established early and the patient is followed carefully to the conclusion of the treatment. Studies being conducted in prevention of the lesion will probably render the presently used operative treatment relatively obsolete. Prophylaxis is simple in concept, applying the concept is extremely difficult and requires close cooperation between physician and nursing staff.

TRADE AND GENERIC NAMES OF DRUGS

Doriden® ..... glutethimide

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BIOPSY BEFORE RADICAL TREATMENT—USEFUL AS A GUIDE TO BREAST CANCER THERAPY

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"The results suggest that a simple excision of the breast lump would prove to be a useful clinical method of sorting out patients with a favorable prospect. The period of delay could be used to advantage for clinical and histological assessment to provide a better prognostic index for each individual patient. Certainly the prevalent concept that a previous excision is a hazard to survival in breast cancer becomes a myth. After the primary breast lump which proves to be carcinoma has been removed, there is a much larger choice of major therapy."

—M. VERA PETERS, M.D., Toronto

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